

Nonperturbative heterogeneous mean-field approach to epidemic spreading in complex networksSergio Gómez,¹ Jesús Gómez-Gardeñes,^{2,3} Yamir Moreno,^{3,4,5} and Alex Arenas^{1,3}¹*Departament d'Enginyeria Informàtica i Matemàtiques, Universitat Rovira i Virgili, E-43007 Tarragona, Spain*²*Department of Condensed Matter Physics, University of Zaragoza, E-50009 Zaragoza, Spain*³*Institute for Biocomputation and Physics of Complex Systems (BIFI), University of Zaragoza, E-50018 Zaragoza, Spain*⁴*Department of Theoretical Physics, Faculty of Sciences, University of Zaragoza, E-50009, Zaragoza, Spain*⁵*Complex Networks and Systems Lagrange Lab, Institute for Scientific Interchange, Viale S. Severo 65, I-10133 Torino, Italy*

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Since roughly a decade ago, network science has focused among others on the problem of how the spreading of diseases depends on structural patterns. Here, we contribute to further advance our understanding of epidemic spreading processes by proposing a nonperturbative formulation of the heterogeneous mean-field approach that has been commonly used in the physics literature to deal with this kind of spreading phenomena. The nonperturbative equations we propose have no assumption about the proximity of the system to the epidemic threshold, nor any linear approximation of the dynamics. In particular, we first develop a probabilistic description at the node level of the epidemic propagation for the so-called susceptible-infected-susceptible family of models, and after we derive the corresponding heterogeneous mean-field approach. We propose to use the full extension of the approach instead of pruning the expansion to first order, which leads to a nonperturbative formulation that can be solved by fixed-point iteration, and used with reliability far away from the epidemic threshold to assess the prevalence of the epidemics. Our results are in close agreement with Monte Carlo simulations, thus enhancing the predictive power of the classical heterogeneous mean-field approach, while providing a more effective framework in terms of computational time.

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I. INTRODUCTION

During the past decade, the physicists' community working on the theory of complex networks has paid special attention to the problem of epidemic spreading in social [1], biological [2–5], and technological networks [6]. The development of mathematical and computational models to guide our understanding of the disease dynamics has allowed one to address important issues, such as the influence of diverse contact patterns and also new algorithms for immunization and vaccination policies [2,7,8]. Physicists' approaches to problems in epidemiology invoke statistical physics, the theory of phase transitions, and critical phenomena [9], to grasp the macroscopic behavior of epidemic outbreaks [1,10–16]. It is not adventurous to claim that one of the main artifices of this success has been the mean-field (MF) approximation, where homogeneity and isotropy are hypothesized to reduce the complexity of the system under study.

On the other hand, the study of the topological properties of complex networks [17–19] has provided new grounds to the understanding of contagion dynamics. Particularly widespread in nature are heavy tailed degree distributions, specially scale-free (SF) networks, whose degree distribution follows a power law $P(k) \sim k^{-\gamma}$ for the number of connections, k , an individual has. SF networks include patterns of sexual contacts [20], the Internet [6], as well as many other social, technological, and biological networks [21]. The critical properties of an epidemic outbreak in SF networks were addressed using the heterogeneous mean-field (HMF) prescription (also called correlated mean field) [1,10–15]. HMF coarse grains vertices within degree classes and hypothesizes that all nodes in a degree class have the same dynamical properties; the approach also assumes that fluctuations can be neglected [10]. This framework has been proved to be exact in annealed networks,

whose nodes' degrees are sampled from a fixed degree distribution at each step of the dynamics [18] (i.e., its specific connectivity is fixed only in average). However, in specific realizations of a model's topology (quenched networks), HMF can result in different levels of accuracy [22]. Moreover, the effect of the topology on the susceptible-infected-susceptible (SIS) epidemic threshold can deviate strongly from HMF [23]. This problem leads to the question of whether or not the direct use of the HMF approach is accurate enough when dealing with real networks (i.e., in an instance of a network ensemble). In a recent work, Gleeson *et al.* [24] studied how accurate the HMF can be on 21 real-world networks and found some relationships between the predictive accuracy and topological properties of the networks. Also, other works have addressed this problem using pair approximations, in particular Miller [25] has found analytical results for a special class of clustered networks.

Although the HMF approach has been extremely useful to assess the critical properties of epidemic models, it is not thought to give information of individual nodes, but of classes of nodes of a given degree. Then, asking about the probability that a given node is infected is not well-posed in this framework. To obtain more details at the individual (node) level of description, usually one has to rely on Monte Carlo (MC) simulations of the actual dynamics, which have also been used to validate the results obtained using HMF methods. The current theory concentrates in two specific situations, the contact process [26–31] (CP) and the fully reactive process [32–34] (RP). A CP stands for a dynamical process that involves an individual stochastic contagion per infected node per unit time, while in the RP there are as many stochastic contagions per unit time as neighbors a node has. However, in real situations, the number of stochastic contacts per unit time is surely a variable of the problem itself [35].

Recently, some of us have proposed an alternative approach to study the susceptible-infected-susceptible (SIS) model [3–5] considering the number of stochastic contacts as a parameter that defines a whole family of SIS models [36], the so-called microscopic Markov-chain approach (MMCA). This more realistic scenario allows one to characterize the prevalence of the disease at the level of individual nodes when the number of contacts interpolates between the two limiting cases of CP and RP. Capitalizing on the MMCA framework, we propose here a nonperturbative HMF formulation which does not prune the equations at first order in the prevalence density, but considers the whole series expansion. The resulting equations can be solved using fixed-point iteration, and are accurate in predicting the epidemic incidence for the whole phase diagram, even out of the critical transition region.

II. DISCRETE-TIME AND CONTINUOUS-TIME SIS MODELS IN NETWORKS

The analysis of epidemic spreading models in networks has some of the same difficulties that are found for well-mixed populations (equivalent to complete graphs). These difficulties are intrinsic to the discrete-time or continuous-time formulation of the governing equations, and the methods used to solve each of them. Continuous approximations have been more popular in epidemic modeling because of their mathematical tractability, and the avoidance of chaotic behaviors that can arise in their discrete counterparts [37]. For the sake of clarity, we henceforth fix our attention on the study of a family of SIS models. In a SIS model, individuals that are cured do not develop permanent immunity but are immediately susceptible to the disease again. In well-mixed populations, the differential equations governing the number of susceptible (S) and infected (I) individuals are

$$\begin{aligned}\frac{dS}{dt} &= -\tilde{\beta}S\frac{I}{N} + \tilde{\mu}I, \\ \frac{dI}{dt} &= \tilde{\beta}S\frac{I}{N} - \tilde{\mu}I,\end{aligned}\quad (1)$$

where $N = S(t) + I(t)$ is the (constant) size of the population. The term I/N accounts for the probability of contacting an infected individual in a well-mixed population of size N , $\tilde{\beta}$ is the infectivity rate (probability per unit time) for each contact, and $\tilde{\mu}$ is the rate at which one infected individual recovers. Their corresponding difference equations are

$$\begin{aligned}S(t + \Delta t) &= S(t) \left(1 - \tilde{\beta}\Delta t \frac{I(t)}{N}\right) + \tilde{\mu}\Delta t I(t), \\ I(t + \Delta t) &= I(t) \left[1 - \tilde{\mu}\Delta t + \tilde{\beta}\Delta t \frac{I(t)}{N} S(t)\right],\end{aligned}\quad (2)$$

or, equivalently,

$$I(t + \Delta t) = I(t) - \tilde{\mu}\Delta t I(t) + \tilde{\beta}\Delta t \frac{I(t)}{N} [N - I(t)].\quad (3)$$

Defining $\rho(t) = I(t)/N$ as the fraction of infected individuals in the population, Eq. (3) is written as

$$\rho(t + \Delta t) = \rho(t) - \tilde{\mu}\Delta t \rho(t) + \tilde{\beta}\Delta t \rho(t)[1 - \rho(t)].\quad (4)$$

Note that, while the system of Eqs. (1) always converges to a solution [37,38], Eq. (4) can be mapped to a logistic

function when the reproductive ratio $\mathcal{R} = \tilde{\beta}/\tilde{\mu} > 1$, thus giving rise to basic periodicity, bifurcations, and chaotic behavior depending on the parameters [37]. Although both descriptions are equivalent in the limit $\Delta t \rightarrow 0$, differences arise when considering a finite Δt .

Particularly interesting is what happens when considering a numerical scheme iterating Eq. (4). In many cases, Δt is usually assimilated to the stochastic simulation time unit and set to 1. Consequently, the numerical differences with the continuous case are substantial. It is also important to distinguish between rates and probabilities: $\tilde{\beta}\Delta t = \beta$ is a probability, and the same holds for $\tilde{\mu}\Delta t = \mu$. Again, by setting $\Delta t = 1$, one can mix up rate and probabilities because both will have the same values, even though their units are different.

The mapping of the above SIS equations to the case of heterogeneous networks is not straightforward and has its critical step in the redefinition of the probability of contacts. In a network, the number of contacts is restricted to a fixed neighborhood, then each individual (node) can potentially contact only its neighbors. In the seminal work by Pastor-Satorras and Vespignani [10], these authors proposed the direct use of Eq. (4) for classes of nodes based on their degree k . This is the root of the HMF approach in complex networks and the hypothesis of homogeneity is here postulated at the level of classes of nodes. The rationale behind this assumption is that the dynamical behavior of any two nodes with the same degree k will be essentially the same. Then, a system of equations for each class k is written as

$$\rho_k(t + \Delta t) = \rho_k(t) - \tilde{\mu}\Delta t \rho_k(t) + \tilde{\beta}\Delta t \Theta_k(t)[1 - \rho_k(t)],\quad (5)$$

where now $\rho_k(t)$ stands for the fraction of infected individuals of degree k , and the probability of contacting an infected node is encoded in the new function $\Theta_k(t)$. For the general case of correlated networks, the function $\Theta_k(t)$ takes the form

$$\Theta_k(t) = \sum_{k'} P(k'|k)\rho_{k'},\quad (6)$$

where $P(k'|k)$ is the probability that a node of degree k connects to a node of degree k' . Equation (5) is used to find the stationary value of the incidence for given values of β and μ . Indeed, for the stationary state, it is true that the only dependence to take into account is the ratio $\lambda = \beta/\mu$ because the equations can be rescaled without modifying their solutions. However, this is not anymore the case during the transient. The critical value λ_c for the epidemic threshold was found in [39] to be

$$\lambda_c = \frac{1}{\Lambda_{\max}(C)},\quad (7)$$

with $\Lambda_{\max}(C)$ being the largest eigenvalue of the connectivity matrix of classes of nodes C , whose components are given by $C_{kk'} = kP(k'|k)$, i.e., the expected number of links from a node of degree k to nodes of degree k' .

III. MICROSCOPIC MARKOV-CHAIN APPROACH TO SIS MODELS

In this section, we summarize an alternative approach to describe the equations governing the SIS class of models [36]. We henceforth refer to this formulation as microscopic

Markov-chain approach (MMCA). The main advantage of this approach is that it is able to deal with the infection dynamics at the level of single nodes. Specifically, we focus on the probability of a node to be infected at time t . For each node i , we construct a Markov chain that accounts for the probability of infection $p_i(t)$, assuming that the number of contacts of node i with its neighbors is parametrized by an integer η , and that the infection events are uncorrelated. Note that this constitutes a first-principles derivation of a discrete model, not a discretization of a differential equation. We consider two different situations of interest: (i) without reinfections (WOR), and (ii) with reinfections (WIR). The first case implies that the time scales for the infection and cure are well separated, whereas the latter assumes that the same time scale holds for infection and cure and therefore a just recovered individual might catch the disease again within the same time step t .

The respective equations are

WOR:

$$p_i(t+1) = [1 - p_i(t)][1 - q_i(t)] + (1 - \mu)p_i(t), \quad (8)$$

WIR:

$$p_i(t+1) = [1 - q_i(t)] + (1 - \mu)p_i(t)q_i(t), \quad (9)$$

where the probability $q_i(t)$ of node i not being infected by any neighbor is

$$q_i(t) = \prod_{j=1}^N [1 - \beta r_{ji} p_j(t)]. \quad (10)$$

Keeping in mind the separation of the two processes, namely, contacting a node and transmitting the infection, already presented in Eqs. (2) for the well-mixed case, the explanation of these equations is straightforward. The terms in the right-hand side of Eq. (8) account respectively for the probability that a susceptible node $[1 - p_i(t)]$ is infected by at least one neighbor $[1 - q_i(t)]$, and an infected node does not recover $[(1 - \mu)p_i(t)]$. Equation (9) adds, after some algebra, a term that accounts for the probability that an infected node recovers $[\mu p_i(t)]$ but gets infected again by a neighbor $[1 - q_i(t)]$ in the same time step. Finally, in Eq. (10), we have the probabilities that infected nodes $[p_j(t)]$ contact node i , and that these contacts lead to new infections, which occur with probability β . The values of the contact probabilities r_{ji} can be expressed as

$$r_{ji} = R_\eta \left(\frac{w_{ji}}{w_j} \right) \quad (11)$$

for weighted networks, and

$$r_{ji} = R_\eta \left(\frac{a_{ji}}{k_j} \right) = a_{ji} R_\eta \left(\frac{1}{k_j} \right) = a_{ji} R_\eta(k_j^{-1}) \quad (12)$$

for unweighted networks, where

$$R_\eta(x) = 1 - (1 - x)^\eta. \quad (13)$$

For the contact process, $\eta = 1$ and $R_1(x) = x$, whereas for the fully reactive process, $\eta \rightarrow \infty$ and $R_\infty(x) = 1$.

At the stationary state, Eqs. (8) and (9) are independent of the discrete time step, and simplify to

$$\text{WOR: } p_i = (1 - p_i)(1 - q_i) + (1 - \mu)p_i, \quad (14)$$

$$\text{WIR: } p_i = (1 - q_i) + (1 - \mu)p_i q_i, \quad (15)$$

with

$$q_i = \prod_{j=1}^N (1 - \beta r_{ji} p_j). \quad (16)$$

These equations are easily solved by fixed-point iteration until a fixed point (for the WIR and WOR cases) or a cycle (for the WOR setup) is found. In this second case, averaging over the oscillating values of the quantity of interest must be considered. Finally, the average fraction of infected nodes in the stationary state is given by

$$\rho = \frac{1}{N} \sum_{i=1}^N p_i. \quad (17)$$

IV. NONPERTURBATIVE HMF

In heterogeneous mean-field theory, it is supposed that all nodes of the same degree behave equally. In terms of the MMCA formulation this means that $p_i = p_j$ if $k_i = k_j$, and the density ρ_k of infected nodes of degree k is given by

$$\rho_k = \frac{1}{N_k} \sum_{j \in K} p_j = p_i, \quad \forall i \in K, \quad (18)$$

where K is the set of nodes with degree k , whose cardinality is denoted by N_k . This notation allows one to group together terms according to the degrees of the nodes. For instance, if the degree of node i is $k_i = k$, then

$$\begin{aligned} \sum_j a_{ji} p_j &= \sum_{k'} \sum_{j \in K'} a_{ji} \rho_{k'} = \sum_{k'} \rho_{k'} \sum_{j \in K'} a_{ij} \\ &= \sum_{k'} \rho_{k'} C_{kk'} = k \sum_{k'} P(k'|k) \rho_{k'}, \end{aligned} \quad (19)$$

where $C_{kk'} = k P(k'|k)$ is the expected number of links from a node of degree k to nodes of degree k' , as explained in Sec. II.

Substitution of the HMF approximation, Eq. (18), into the MMCA Eqs. (14) and (15) leads to

$$\text{WOR: } \rho_k = (1 - \rho_k)(1 - q_k) + (1 - \mu)\rho_k, \quad (20)$$

$$\text{WIR: } \rho_k = (1 - q_k) + (1 - \mu)\rho_k q_k, \quad (21)$$

which can also be written as

$$\text{WOR: } 0 = -\mu\rho_k + (1 - \rho_k)(1 - q_k), \quad (22)$$

$$\text{WIR: } 0 = -\mu\rho_k + (1 - (1 - \mu)\rho_k)(1 - q_k). \quad (23)$$

These equations constitute the HMF approximations of MMCA for the SIS model without and with reinfections, respectively.

We still need a HMF expression for q_k . For unweighted networks, the value of q_i is

$$q_i = \prod_{j=1}^N (1 - \beta r_{ji} p_j) = \prod_{j=1}^N [1 - \beta a_{ji} R_\eta(k_j^{-1}) p_j]. \quad (24)$$

If node i has degree $k_i = k$, then

$$q_i = q_k = \prod_{k'} \prod_{j \in K'} [1 - \beta a_{ji} R_\eta(k'^{-1}) \rho_{k'}], \quad (25)$$

where we have grouped together the terms in the product by their degrees k' as in Eq. (19). The expression within the parentheses is equal to 1 for all nodes of degree k' that are not connected to node i ($a_{ji} = 0$), and equal to $[1 - \beta R_\eta(k'^{-1}) \rho_{k'}]$ for nodes of degree k' that are linked to i ($a_{ji} = 1$). Besides, the expected number of such terms is $C_{kk'}$. Hence we obtain

$$q_k = \prod_{k'} [1 - \beta R_\eta(k'^{-1}) \rho_{k'}]^{C_{kk'}}. \quad (26)$$

Equations (22) and (23), together with Eq. (26), form what we call the nonperturbative heterogeneous mean-field (npHMF) equations of the SIS model in unweighted networks. Note that in the derivation of the npHMF equations no assumption has been made about the proximity of the system to the epidemic threshold, where the epidemic prevalence is small, nor any linear approximation has been invoked; hence the qualification of “nonperturbative.”

The solution of the npHMF equations follows the same steps as in MMCA, i.e., Eqs. (20), (21), and (26) are iterated until a fixed point (WIR, WOR) or a cycle (WOR) is found. As before, for the latter case, we take the average of oscillating values for the disease prevalence. Finally, the global epidemic prevalence is given by

$$\rho = \frac{1}{N} \sum_k N_k \rho_k. \quad (27)$$

It is easy to show that the standard HMF equations [31,39] are just a linear approximation of our WOR npHMF equations. Near the epidemic threshold β_c , where $\rho_k \ll 1$, we get

$$\begin{aligned} q_k &\sim 1 - \beta \sum_{k'} C_{kk'} R_\eta(k'^{-1}) \rho_{k'} \\ &= 1 - \beta k \sum_{k'} P(k'|k) R_\eta(k'^{-1}) \rho_{k'}, \end{aligned} \quad (28)$$

which can be inserted into Eq. (22) to give

$$0 = -\mu \rho_k + \beta k (1 - \rho_k) \sum_{k'} P(k'|k) R_\eta(k'^{-1}) \rho_{k'}, \quad (29)$$

where

$$R_\eta(k'^{-1}) = \begin{cases} 1 & \text{for the RP,} \\ \frac{1}{k'} & \text{for the CP.} \end{cases} \quad (30)$$

A. Epidemic threshold

To round off our analysis, we derive the critical spreading threshold using the different approaches discussed here. In [36], it is shown that MMCA allows the determination of the epidemic threshold,

$$\beta_c = \frac{\mu}{\Lambda_{\max}(R)}, \quad (31)$$

where $\Lambda_{\max}(R)$ is the maximum eigenvalue of the matrix R of the contact probabilities r_{ij} . In particular,

$$\beta_c = \begin{cases} \frac{\mu}{\Lambda_{\max}(A)} & \text{for the RP,} \\ \mu & \text{for the CP.} \end{cases} \quad (32)$$

These results hold both with and without reinfections, since at first order, Eqs. (8) and (9) coincide. In the same way, the critical points from npHMF Eqs. (22) and (23) are the same as in standard HMF, where the matrix H with elements $h_{kk'} = C_{kk'} R_\eta(k'^{-1})$ replaces matrix R ,

$$\beta_c^{\text{HMF}} = \frac{\mu}{\Lambda_{\max}(H)}, \quad (33)$$

with the well-known particular cases,

$$\beta_c^{\text{HMF}} = \begin{cases} \frac{\mu}{\Lambda_{\max}(C)} & \text{for the RP,} \\ \mu & \text{for the CP.} \end{cases} \quad (34)$$

The comparison with Monte Carlo (MC) simulations shows that MMCA provides a better approximation to the epidemic threshold than HMF, since no information of the original network structure is lost. Additionally, by the definition of the contact probabilities, the MMCA is applicable to weighted and directed networks without further modifications of the equations. This is not the case when one deals with HMF in networks that are not unweighted and undirected.

V. COMPARISON WITH MONTE CARLO SIMULATIONS

To compare the results coming out from the different approaches, we have performed MC simulations of the disease dynamics on top of scale-free networks generated using the uncorrelated configuration model. MC simulations are performed scheduling the dynamics from the nodes, not from the links. At every simulation step, the epidemics dynamics updates synchronously the state of all the nodes. Besides, numerical solutions of the fixed-point equations have also been obtained. For high values of μ and β , MMCA without reinfections does not converge to a fixed point as the rest, but it converges to an oscillation between two states, from which the average is taken. These oscillations are also present in the MC runs, and disappear after averaging over multiple runs.

Standard HMF cannot be solved by iteration, since it diverges even for small values of $\beta \sim 10^{-1}$. Therefore, we have made use of a nonlinear minimization algorithm based on a successive quadratic programming solver, which is a gradient-based optimization routine based upon the method of Lagrange multipliers to incorporate the inequality constraints. More precisely, we have minimized the error function,

$$\sum_k (-\mu \rho_k + \beta k (1 - \rho_k) \sum_{k'} P(k'|k) R_\eta(k'^{-1}) \rho_{k'})^2, \quad (35)$$

with the constraints $0 \leq \rho_k \leq 1$ using the “sqp” function in *Octave* [40]. Since the number of different degree classes is much smaller than the size of the network (e.g., 55 different degrees for our $N = 10^4$ network in Fig. 1), the algorithm is able to solve the problem in a reasonable amount of time.

Figure 1 shows the comparison between all the approaches discussed throughout this paper for the case in which a fully reactive process is considered. As can be seen, the worst

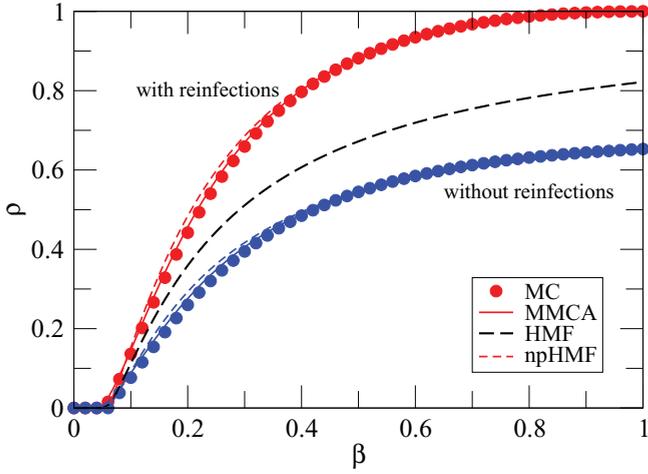


FIG. 1. (Color online) Epidemic prevalence ρ as a function of the infection rate β . The different curves correspond (as indicated) to Monte Carlo results and the numerical solutions of the fixed-point equations of the different approaches (MMCA, HMF, and npHMF) for a SF network made up of $N = 10^4$ nodes using the configuration model. The exponent of the degree distribution is $\gamma = 2.7$. The contact probabilities used correspond to the case of a fully reactive process and μ has been set to 0.5.

performance with respect to MC simulations corresponds to the HMF, which correctly predicts the epidemic threshold, but fails to reproduce the evolution of the epidemic incidence as the infection rate increases and moves away from the critical point. The figure also shows that the npHMF approximation behaves only slightly worse than the Markov-chain formulation. More important, in addition to correctly capturing the critical epidemic threshold and at variance with the standard HMF, it allows one to study the whole phase diagram, whatever the value of the infection rate is.

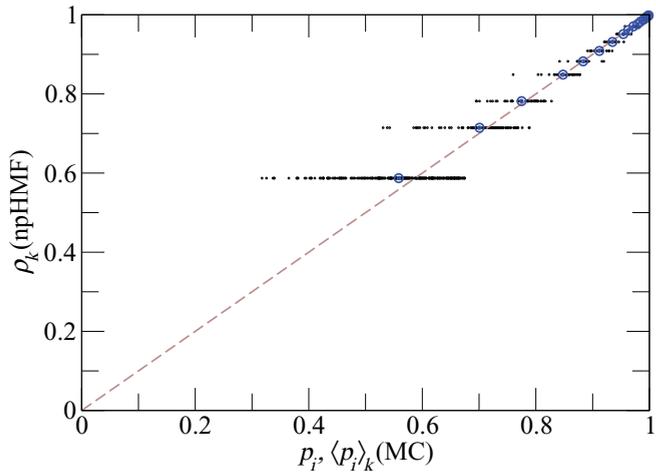


FIG. 2. (Color online) Fraction ρ_k of infected individuals of degree k from npHMF vs the infection probabilities p_i (black) and their average $\langle p_i \rangle_k$ over nodes with the same degree (blue) obtained from MC. The epidemic model is a RP with reinfections with $\mu = 0.5$ and $\beta = 0.3$. For the sake of clarity, we have used in this case an SF network of 500 nodes with $\gamma = 2.7$.

To provide further evidences of the validity of the npHMF, we have represented in Fig. 2 the epidemic incidence of nodes of degree ρ_k as given by the solution of the npHMF equations as a function of the probability p_i that a node i , whose connectivity is k , is infected, with the latter being obtained from MC simulations. Besides, the average over all the p_i 's for nodes of the same degree has also been represented. As can be seen, all individual probabilities are distributed around the mean value, which in turn is close to the values coming out from the npHMF formulation. These results further illustrate that the behavior is not the same along all the connectivity classes: for large prevalence densities (or probability of being infected), the dispersion around the mean values shrinks. In other words, the degree of accuracy in the prediction of the state of a given node i with respect to whether it is infected or not depends on ρ_k : for large values of this density, one can

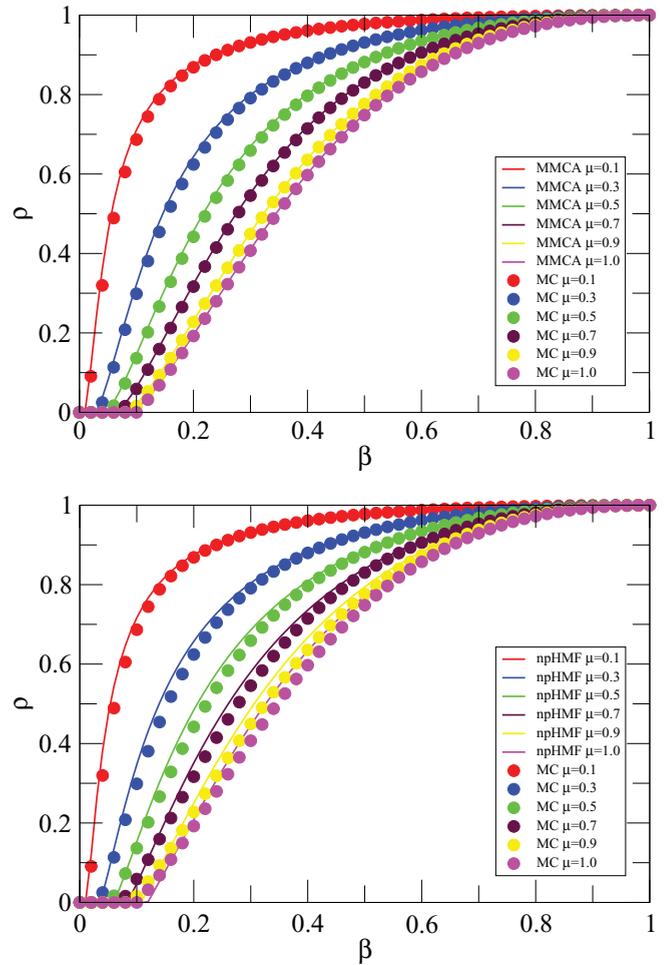


FIG. 3. (Color online) Epidemic prevalence ρ as a function of the infection rate β for increasing values of μ from left ($\mu = 0.1$) to right ($\mu = 1.0$). The top panel corresponds to the numerical solutions of the fixed-point equations of the MMCA formulation, while the bottom figure has been obtained using the npHMF equations. In both cases, the numerical solutions are compared with the results of MC simulations on top of a SF network made up of $N = 10^4$ nodes. The exponent of the degree distribution is $\gamma = 2.7$. The contact probabilities used correspond to the case of a fully reactive process.

predict with high accuracy the individual probabilities p_i for nodes of degree k , while if ρ_k is relatively small, the prediction error is larger due to the more pronounced deviations from the average value.

Finally, we have also tested the new formulation against variation of the recovery rate. The results obtained for different values of μ using the MMCA and the npHMF approaches are compared with MC simulations in Fig. 3. As in Fig. 1, we have prescribed a fully reactive process on top of an undirected and unweighted scale-free network. Moreover, the results shown correspond to the case in which reinfections are possible (similar results are found for the case without reinfections). The different curves show that both formulations perform quite well, with, however, the MMCA being slightly better than the npHMF case.

VI. CONCLUSIONS

In summary, in this paper we have proposed a new kind of heterogeneous mean-field approach to describe the spreading of diseases in complex networks. Capitalizing on a previous formulation aimed at computing the individual probabilities for nodes to be infected, we have coarse grained the dynamical equations by degree classes. In doing so, we have been able to keep higher-order terms, which ultimately allow us to use the resulting fixed-point equation to obtain numerical solutions that are in good agreement with MC simulations. However, although the results outperform those obtained through the standard mean-field approach, the new formulation is still

limited with respect to individual-based approaches (such as the MMCA). The reason is that, when coarse graining the dynamics, some information of the original network is lost and exact quantities are replaced by expected values. The ultimate consequence is that, although one can accurately reproduce the behavior of global dynamical descriptors like the epidemic prevalence, at the individual level the probabilities of infection are more error-prone. Having said that, we however think that the approach proposed here represents a significant improvement on the standard HMF framework in several aspects. First, it is more accurate. Secondly, it allows one to solve the whole phase diagram, which opens up the possibility of getting fast numerical solutions given a network topology without resorting to MC simulations.

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